Amendments to the Specification:

Please amend page 4, line 9-16 of the specification as follows:

Thus, in a first aspect the invention features a method of detecting a retroviral genetic recombinant, preferably a recombinant having gag and pol functions which can be complemented by helper function(s) supplied by or to the cell, and which helper function(s) facilitate propagation of the recombinant to permit the recombinant's detection. This detection can take place using any one of a variety of biochemical, diagnostic, and/or functional assays that can identify the recombinant. Preferably, the assay makes use ~~used~~ of the gag and pol functions contained within the recombinant, and a marker gene to facilitate the detection.

Please amend page 5, lines 6-19 as follows:

"Propagating" or "propagated" refers to the ability of the recombinant to integrate and duplicate in the host cell, e.g., as part of a mitosis event, and/or to support viral replication and mobilization, e.g., using various helper functions that are supplied by or to the host cell. "Helper functions" are used to help facilitate propagation of the recombinant when the recombinant is one that is incapable or inefficient at replicating, packaging, and/or infecting by itself. Examples of helper functions include but are not limited to the retroviral env gene product and pseudotypes thereof, and/or the retroviral tat and rev gene products. Depending on the embodiment, various helper functions can be provided individually or in combination. Usually, although not necessarily, these helper functions will originate from a different nucleic acid strand in the cell relative to the recombinant strand, although each may ultimately share the feature of being integrated into the host genome. This is usually a consequence of the "split function" aspect of most retroviral systems that is directed to "disarming" viruses and promoting biological containment and safety.

Please amend page 7, lines 25-29 of the specification as follows:

In embodiments wherein a mobilization is used to determine the presence of the recombinant, that mobilization may be of the recombinant itself and/or of a marker gene. For this, the marker gene preferably has a retroviral packaging sequence, a promoter ~~promter~~, and flanking LTR elements, like the recombinant. The promoter may be an inducible promoter or a constitutive promoter.

Please amend page 10, lines 4-7 of the specification as follows:

The marker ~~marker~~ gene can be a reporter gene or a selective marker gene. Preferably it is a selective marker gene, more preferably an antibiotic resistance gene, and most preferably a puromycin resistance gene. The marker preferably has an expression that is controlled by a promoter that is present, whether inducible or constitutive.

Please amend page 16, lines 11-12 as follows:

Preferably, a step is also incorporated in which the recombinant provirus' presence is determined using an inhibitor of reverse transcriptase ~~transcripase~~, e.g., Nevirapine.

Please amend page 19, lines 24-29 as follows:

In another aspect, the invention features a Gag transfer assay that includes an indicator cell having a selectable marker gene. The selectable marker gene is expressed in response to a retroviral protein encoded by a recombinant provirus. The recombinant provirus includes functional gag and pol genes that are capable of producing functional Gag and Pol proteins. The selectable marker gene encodes a product which can be selected for in a growth media for the cell under a defined environmental condition.

Please amend page 23, lines 26 as follows:

Fig. 27c shows the codon usage of highly expressed human genes.

Please amend page 36, lines 29-30 and page 37, lines 1-10 as follows:

Although it seemed unlikely, it was possible that the *tat* and *gag* genes were transferred without recombination ~~recombination~~ (pseudotransduction), via the pTRE-gag-pol packaging plasmid DNA carried over from the production of the vector stock. To test for this possibility, infections of the HeLa-puro cells were performed in the presence of Nevirapine, a non-nucleoside reverse transcriptase inhibitor specific to the HIV-1 RT. (Figs. 15-17) Nevirapine would prevent only HIV-RT mediated transfer of *tat* and *gag* into the HeLa-puro cell but not if it is transferred via plasmid DNA. These figures show the results of the tat transfer, gag transfer, and marker rescue assays which were performed as described before except that the infection of HeLa-puro was carried out in the presence of 1 ug/ml Nevirapine (NVP). No puromycin-resistant colonies formed in any of the assays, indicating that these transfers were mediated by HIV-1 reverse transcriptase and not through plasmid DNA contamination.

Please amend page 38, lines 23-30 and page 39 as follows:

In order to confirm that the puro resistant colonies that were produced by complementation of the vector particles with the Vpr-RT-IN fusion protein were indeed RT-IN deficient, the resistant cells were expanded in culture and the high molecular weight DNA was extracted for genetic analysis (Fig. 20). This analysis demonstrated that ~~that~~ the RT-IN coding region was defective since 5 of 5 recombinants that were analyzed contained a translational stop codon (TAA) at the first amino acid residue of RT, as introduced into the trans-lentiviral packaging construct originally. By comparison, 5 of 5 recombinants of the lentiviral vector contained an open RT-IN reading frame (Fig. 20).